ANNALS OF SURGERY
Vol. 236, No. 4, 471–479

Thoracoabdominal Aneurysm Repair: Results With 337 Operations Performed Over a 15-Year Interval

Richard P. Cambria, MD,* W. Darrin Clouse, MD,* J. Kenneth Davison, MD,† Peter F. Dunn, MD,† Michael Corey,* and David Dorer, PhD

*From the Divisions of *Vascular Surgery and †Vascular Anesthesia and the Thoracic Aortic Center, Surgical and Anesthesia Services, Massachusetts General Hospital and the Harvard Medical School, Boston, Massachusetts*

Objective

To review perioperative results and late survival after thoracoabdominal aneurysm repair (TAA), in particular to assess the impact over time of epidural cooling (EC) on spinal cord ischemic complications (SCI).

Summary Background Data

A variety of operative approaches and protective adjuncts have been used in TAA to minimize the major complications of perioperative death and SCI. There is no consensus with respect to the optimal approach.

Methods

From January 1987 to November 2001, 337 consecutive TAA repairs were performed by a single surgeon. Clinical features included prior aortic grafts in 97 (28.8%) and emergent operation in 82 (24.6%), including rupture in 46 (13.6%) and dissection in 63 (19%). Operative management consisted of a clamp/sew technique with adjuncts in 93%. EC (since July 1993) to prevent SCI was used in 194 (57.6%) repairs. Variables associated with the end points of operative mortality and postoperative SCI were assessed with the Fisher exact test and logistic regression; late survival was estimated with the Kaplan-Meier method.

Results

Operative mortality was 8.3% and was associated with nonelective operation, intraoperative hypotension, total transfusion requirement, and the postoperative complications of paraplegia, renal failure, and pulmonary insufficiency. Postoperative renal failure and transfusion requirement were independent correlates of mortality. SCI of any severity occurred in 38 of 334 (11.4%) operative survivors, with 22/38 (6.6% of cohort) sustaining total paraplegia. EC reduced the risk of SCI in patients with types I–III TAA (10.6% vs. 19.8%, $P = .04$). Independent correlates of SCI over the entire study interval included types I/II TAA, rupture, cross-clamp duration, sacrifice of T9–L1 intercostal vessels, and intraoperative hypotension. Late survival rates at 2 and 5 years were 81.2 ± 3% and 67.2 ± 5%.

Conclusions

EC has decreased the risk of SCI after TAA repair. Decreasing the substantial proportion (nearly 25%) of patients requiring nonelective operation will improve results. Late survival is equal to that after routine AAA repair, indicating that the considerable resource expenditure required for TAA repair is worthwhile.

Despite advances in endoluminal grafting for isolated thoracic and infrarenal aortic aneurysm, open operation remains the only feasible treatment option for thoracoabdominal aneurysm (TAA). Since TAA extent is relatively uncommon in the spectrum of degenerative aneurysm disease, experience and clinical reports have been largely concentrated in a modest number of centers. Even in such environments, operative mortality is generally in the 10%

Presented at the 122nd Annual Meeting of the American Surgical Association, April 24–27, 2002, The Homestead, Hot Springs, Virginia.
Supported in part by the Harold and June Geneen Vascular Surgery Research Fund.
Correspondence: Richard P. Cambria, MD, Massachusetts General Hospital, 15 Parkman Street WAC 458, Boston MA 02114
E-mail: rcambria@partners.org

Accepted for publication April 24, 2002.
DOI: 10.1097/01.SLA.0000031240.26001.E2

range, and spinal cord ischemic complications (SCI) remain in the 5% to 15% range.[1–9] Indeed, efforts to minimize the risk of SCI have been the principal impetus for the variety of operative strategies and protective adjuncts used during surgery. Clearly, there is no consensus on the major variants of operative approach (e.g., with or without some form of distal aortic perfusion) designed to minimize SCI, as reviewed elsewhere.[10]

Our approach to TAA repair has emphasized operative simplicity and expediency while relying on adjuncts directed against the principal complications. While detailed in other reports,[11] this strategy essentially uses a clamp/sew technique, regional hypothermia for spinal cord and renal protection,[12,13] and in-line mesenteric shunting to minimize visceral ischemia.[14] Recently, our institutional experience with epidural cooling (EC) for the prevention of SCI was published. Devastating total paraplegia was reduced to the 2% range overall, yet 12% of patients with types I/II TAA still sustained some degree of deficit.[15] In the present report we review our 15-year experience to identify predictors of the hard end points of operative mortality and SCI. In addition, we assess the particular impact of EC (adopted in 1993) on SCI over the study interval.

METHODS

From January 1987 to December 2001, 337 consecutive TAA repairs were performed in 333 patients by the senior author (R.P.C.). Progressive aneurysmal enlargement proximal (two patients) or distal (two cases) to our prior repair (one case with rupture) led to repeat operation in four patients. Aneurysms were classified according to the Crawford scheme, with patients designated extent I/II having a proximal anastomosis immediately adjacent to the left subclavian origin and consequent resection of the entire descending aorta. Isolated descending thoracic aneurysms whose resection could be encompassed with an anastomosis proximal to the celiac axis were omitted. Primary aortic operation was carried out in 72% of the cohort, secondary in 24%, and tertiary in 1.5%. Clinical and demographic features are detailed in Table 1. Note the essentially one-to-one male-to-female sex ratio. Pulmonary function studies are routinely obtained and were the basis for quantification of COPD, as detailed in Table 1. Urgent operation was defined as either rupture or presentation with back/abdominal pain necessitating observation in an intensive care unit and operation within 48 hours of admission.

Operation was carried in most (93%) patients with a clamp/sew technique with adjuncts; distal aortic perfusion techniques were used in highly selected circumstances, such as the anticipation of technical complexity in construction of the proximal anastomosis (e.g., chronic dissection) or in patients with significant renal insufficiency. EC has been used for spinal cord protection since July 1993 (types I–III TAA) and was supervised in all cases by a dedicated vascular anesthesia team (J.K.D., P.F.D.). Details of the epidural infusion system have been previously published.[12] Patent intercostal vessels in the T9-L1 region were reimplanted by means of a separate inclusion button or were preserved with beveled anastomosis when technically feasible. Following reperfusion of the lower extremities, EC was discontinued and continuous passive CSF drainage initiated (since 1999).

Table 1. DEMOGRAPHIC AND CLINICAL DATA

	Mean ± SD	n (%)
Age (years)	70.5 ± 9.2	
Gender (male)		160 (48)
Diabetes mellitus		24 (7.1)
Hypertension		294 (87.2)
Family aneurysm history		32 (9.5)
Serum Cr ≥1.8 mg/dL		43 (12.8)
CAD (by history or ECG)		180 (53.4)
COPD		191 (56.7)
Mild		93 (27.6)
Moderate		45 (13.4)
Severe		53 (15.7)
Aneurysm pathology		
Degenerative		264 (78.3)
Dissection		63 (18.7)
Infected		5 (1.5)
Aortitis		5 (1.5)
TAA extent		
I		92 (27)
II		59 (17)
III		120 (36)
IV		66 (20)
Prior aortic resection (no. patients)		97 (28.8)
All prior aortic operations (n = 110)		
AAA		61 (62.9)
Asc/arch		25 (25.8)
Desc Ao/TAA		24 (24.7)
Clinical presentation		
Elective		255 (75.7)
Urgent nonruptured		36 (10.7)
Ruptured		46 (13.6)

Operative mortality was defined as any death within 60 days of operation or any other death occurring during the initial hospitalization. All patients were awakened in the operating room for an initial neurological examination of the lower extremities. SCI were classified as immediate when noted as the patient awoke from anesthesia, or as delayed when patients were initially neurologically intact. Cardiac complications included documented MI, arrhythmia requiring therapy, CHF requiring therapy, or unstable angina. Major pulmonary complications included mechanical ventilation for more than 72 hours postoperatively, reintubation or transfer back to ICU for respiratory failure, documented pneumonia, or the need for tracheostomy. Significant renal failure consisted of need for dialysis; postoperative creatinine levels of 3.0 mg/dL or more (in patients with normal baseline renal function); or doubling of base-

Table 2. INTERCOSTAL ARTERY MANAGEMENT Segmental Intercostal Artery Salvage in 337 Thoracoabdominal Aortic Aneurysm Repairs

	Type I (n = 92)	Type II (n = 59)	Type III (n = 120)	Type IV (n = 66)
IC reattachment [patients, n (%)]*	63 (69%)	39 (66%)	61 (51%)	22 (33%)
Time to antegrade IC reperfusion (min ± SD)	40.1 ± 21.4	48.8 ± 23.6†	31.9 ± 13.7	30.9 ± 9.5
% of IC levels reattached in T_9-L_1 zone	80.0%	82.5%	90.3%	100%

* Reattachment = inclusion button, bypass graft or preservation in beveled anastomosis.
† Type II reperfusion time significantly greater than type III ($P = .001$) and type IV ($P = .01$).

line creatinine for those with baseline creatinine at least 1.8 mg/dL.

Data were entered into an Excel for Windows (v97) spreadsheet (Microsoft, Inc., Redmond, WA). Data were transferred to a statistical package software and univariate, multivariate, and survival analyses performed. Patients who died in the operating room (n = 3) were eliminated for the purpose of calculating complication incidence. Intraoperative variables were examined and reported as mean ± SD. Comparisons were performed between groups using unpaired Student *t* tests, and comparisons of multiple groups were made by analysis of variance with posthoc testing. The influences of dichotomous variables on the two outcomes of SCI and in-hospital death were determined using the Fisher exact test for two-by-two contingency tables. The odds ratios reported for the dichotomous variables are based on the conditional maximum likelihood estimate. Continuous variables were compared for the two groups using the Wilcoxon rank-sum test for unmatched samples. Multivariate logistic regression models were constructed by including all variables that were significant at the $P = .05$ level. Adjusted odds ratios were reported. Postoperative survival time was determined using a Kaplan-Meier estimate.

RESULTS

Visceral cross-clamp time averaged 10 minutes longer in extent I/II TAA ($P < .0001$) because of the additional time required for inclusion anastomosis of the intercostal vessels (performed in 43% of type I/II repairs in this cohort) and because many type IV reconstructions are carried out with a single beveled anastomosis to both the visceral segment and the proximal aorta. Specific management of intercostal vessels is detailed in Table 2. These data reflect an aggressive posture toward intercostal vessel preservation/reconstruction. Nearly 70% of type I/II patients had some intercostals reconstructed, the majority of these (≈80%) in the critical T_9-L1 segment.

Operative mortality was 8.3% (elective cases 17/255 [6.7%], urgent cases 11/82 [13.4%], $P = .04$). There were three intraoperative deaths, two in the setting of ruptured TAA and hemorrhage and one from coagulopathic hemorrhage. Preoperative, intraoperative, and postoperative variables correlated with operative mortality are displayed in Table 3. As expected, a dominant influence on mortality related to the major postoperative complications of SCI and renal failure, the latter retaining significance as an independent predictor of early death after multivariate analysis (OR 6.9, 95% CI 2.7–17.9, $P = .00006$). The only other variable independently associated with perioperative mortality was total transfusion requirement (OR 1.4, 95% CI 1.1–1.7, $P = .005$). Nonfatal complications of operations are displayed in Table 4. The incidence of renal failure was 15/81 (18.5%) in urgent cases versus 30/253 (11.9%) in elective cases ($P = .13$). SCI of any severity occurred in 38/334 (11.4%) of patients, with 22 of these 38 (58% of deficits, 6.6% of cohort) sustaining total paraplegia. Seventy percent of deficits were immediate, with the remainder occurring in delayed fashion hours to even weeks postoperatively. The overall incidence of any degree of lower extremity neurological deficit stratified according to TAA extent and mode of presentation is detailed in Table 5. Overall, SCI occurred more frequently in extent I/II (vs. III/IV) TAA (26/151 [17.2%] vs. 12/186 [6.5%], $P = .002$). Univariate and multivariate analyses of variables associated with SCI are displayed in Tables 6 and 7. Use of EC (since July 1993) in type I–III TAA was associated with a significant reduction in SCI (19/180 [10.6%] vs. 18/91 [19.8%], $P = .04$, OR 2.1).

Duration of late follow-up was a mean of 24 months (interquartile range 2.7–38.4 months). As displayed in Figure 1, actuarial survival rates at 2 and 5 years were 81.2 ± 3% and 67.2 ± 5%, respectively.

DISCUSSION

Despite the evident and appropriate focus on SCI, a review of contemporary reports indicates that perioperative mortality remains a significant issue in TAA repair. Representative large clinical series, including the most recent publications, indicate that the mortality of TAA repair remains in the 10% range, identical to Crawford's series, which detailed patients treated between 1960 and 1991[1] (Table 8). A consideration of variables predictive of operative mortality reveals consistent themes. First, mortality in circumstances other than elective operation is essentially doubled. While perhaps intuitive, this factor is particularly

Table 3. UNIVARIATE ANALYSIS OF VARIABLES EXAMINED FOR ASSOCIATION WITH PERIOPERATIVE DEATH

	In-Hospital Death		
	No (n = 309) n (%)	Yes (n = 28) n (%)	*P* Value (OR; 95% CI)
Preoperative			
Age ≥70 yrs	194 (63)	19 (66)	.84
Male	144 (47)	17 (59)	.25
Serum Cr ≥1.8 mg/dL	42 (14)	8 (28)	.06
COPD (severe)	47 (15)	6 (21)	.43
TAA type I-II (n = 151)	139 (45)	12 (43)	.62
Urgent/emergent operation	70 (23)	12 (41)	.04 (2.4; 0.99–5.6)
Rupture	38 (12)	8 (28)	.04 (2.7; 0.96–6.9)
Dissection	56 (18)	7 (24)	.46
Intraoperative			
Intraoperative hypotension	37 (12)	13 (45)	.00004 (5.9; 2.4–14.3)
Operative time (min)*	309.9 ± 95.0	334 ± 97.6	.07
Total cross-clamp (min)*	73.9 ± 26.0	81.3 ± 27.7	.25
Visceral cross-clamp (min)*	46.9 ± 13.9	54.7 ± 21.8	.11
Total operative transfusion (cc)*	2570 ± 1,442	5,001 ± 4,254.3	.0002
Postoperative			
Reexploration for bleeding	11 (4.5)	3 (10)	.31
Spinal cord ischemia (any severity)	30 (10)	8 (28)	.009 (3.5; 1.2–9.2)
Major paraplegia	14 (4.5)	8 (28)	.0002 (7.9; 2.6–23.0)
Renal failure	29 (9)	16 (55)	.00000002 (11.6; 4.7–29.3)
Cardiac complications	32 (10)	7 (24)	.06
Pulmonary complications	129 (42)	18 (62)	.05 (2.3; 0.97–5.5)
Stroke	9 (3)	3 (10)	.07

Fisher's exact test used for proportions.
* Wilcoxon (Mann-Whitney) rank-sum test used for continuous variables.
Continuous data: mean ± SD.

relevant in TAA repair since the percentage of patients treated in nonelective circumstances has been in our experience a consistent 20% of cases, far in excess of corresponding figures for AAA repair. The explanation for increased mortality in urgent cases is not so simple as hemodynamic instability associated with rupture, since most of these patients are stable during their preoperative preparation. Nonetheless, the association of nonelective operation with perioperative mortality is verified in this series and others.[1,3,6] Since elective candidates for TAA typically undergo cardiopulmonary profiling, it is possible that patients presenting with impending rupture represent a high-risk subgroup, having been denied elective repair because of associated comorbidities. Although our results have failed to implicate advanced age as increasing perioperative risk, others have reached the opposite conclusion.[3,6] In our view, the patient's overall functional status assumes greater importance than chronologic age. Indeed, Huynh et al have recently reported acceptable perioperative mortality (15%) in 63 octogenarians who underwent TAA repair.[16] The presence of significant cardiopulmonary morbidities would be expected to increase perioperative risk, and often problematic in clinical decision making is the presence and severity of COPD, present to the some degree in 50% of patients. While our data and those of others implicate significant COPD as a risk factor for development of postoperative pulmonary complications,[2] it is also true that COPD represents an independent risk factor for TAA expansion and rupture.[17,18] Balancing these risks is a common dilemma in clinical decision making in patients with TAA. The final consideration in the patient's preoperative profile requiring careful consideration relates to renal dysfunction, present in some 13% of our patients, similar to that reported elsewhere.[1,3,6] Our experience has correlated preoperative renal dysfunction with increased operative mortality,[2] although this association was weak ($P = .06$) in the present series. Alternatively, Coselli et al noted renal dysfunction to be an independent predictor of operative mortality, increasing it more than threefold.[6] It is likely that this effect is related to the often-demonstrated relationship between antecedent renal dysfunction and postoperative renal failure.[1,3,13,19,20] In turn, renal failure complicating TAA repair, irrespective of the requirement for dialysis, has been a consistent and powerful predictor of early mortality throughout our experience and that of others.[1–3] As reported herein, a sevenfold increased risk of postoperative death can be anticipated in those who sustain postoperative renal failure. As previously reviewed, there is no superiority of

Table 4. POSTOPERATIVE COMPLICATIONS IN 337 TAA REPAIRS

Complication	n (%)
Any renal failure*	45 (13.5%)
Dialysis	16 (4.8%)
Cardiac	46 (13.8)
MI	13 (3.9)
Arrhythmia	28 (8.4)
CHF	6 (1.8)
Unstable angina	1 (0.3)
Stroke	12 (3.6)
Pulmonary	147 (44)
Gut ischemia	7 (2)
Reoperation	82 (24.6)
Reexploration for bleeding	17 (5.1)
Tracheostomy	38 (11.4)
Gastrostomy	24 (7.2)
GI ischemia	5 (1.5)
GI (perforation/obstruction/bleeding/suspected sepsis)	12 (3.6)
Renal thrombectomy/bypass	6 (1.8)
Peripheral vascular intervention (thrombectomy/bypass)	6 (1.8)

* See text for definition.

any specific renal protective adjunct (i.e., distal perfusion vs. regional hypothermia).[2] While it is intuitive that the major complications of operation would contribute significantly to in-hospital death, the disproportionate effect of renal failure is striking, particularly with consideration that only one third of these patients have required dialysis in our cohort. The major impact of antecedent renal dysfunction and its relation to postoperative renal failure, accordingly, has important implications for clinical decision making. We carefully assess renal function in the context of associated renovascular occlusive disease, which is present in up to 40% of patients being considered for TAA repair.[2,13] If the potential for concomitant (with TAA repair) correction of flow-limiting renovascular lesions does not exist, then significant (serum creatinine $\geq$ 2.0 mg/dL) baseline renal dysfunction constitutes a relative contradiction to TAA repair. Finally, similar to the dilemma with COPD and its influence on TAA rupture and postoperative complications, some natural history studies have implicated renal failure as increasing the risk of aneurysm expansion and rupture.[17,21]

With regard to SCI after TAA repair, this study confirms the importance of clinical and anatomical variables generally associated with the risk of SCI throughout the history of TAA surgery. Specifically, SCI is concentrated among the more extensive lesions, with type II TAA having the single highest risk (see Table 5), consistent with the results of others.[3,6,8] Similar to its impact on early mortality, operation in nonelective circumstances is accompanied by at least a threefold increase in the risk of SCI. Particularly relevant to our cohort, in which clamp/sew has been the principal operative technique, the influence of cross-clamp duration, as originally emphasized by Svensson et al,[1] remains paramount. This variable becomes both unclear and of perhaps less importance in those centers where distal aortic perfusion methods are routinely used.[22,23] Prospective recording of the intraoperative treatment of intercostal vessels has allowed us to assess the impact of intercostal vessel management. In a prior report focusing on the use of regional hypothermia for spinal cord protection, we noted a 10-fold

Table 5. INCIDENCE OF SPINAL CORD ISCHEMIA IN 337 THORACOABDOMINAL ANEURYSM REPAIRS BY ONSET OF PRESENTATION

Aneurysm Type	Overall	Immediate	Delayed
I (n = 92; 1 operative death)			
(13/91 = 14.3%)			
Major paraplegia	5 (5.5%)	4 (4.4%)	1 (1.1%)
Paraparesis	8 (8.8%)	3 (3.3%)	5 (5.5%)
II (n = 59)			
(13/59 = 22%)			
Major paraplegia	9 (15.3%)	7 (11.9%)	2 (3.4%)
Paraparesis	4 (6.8%)	4 (6.8%)	0 (0%)
III (n = 120; 1 operative death)			
(11/119 = 9.2%)			
Major paraplegia	7 (5.9%)	3 (2.5%)	4 (3.4%)
Paraparesis	4 (3.4%)	4 (3.4%)	0 (0%)
IV (n = 66; 1 operative death)			
(1/65 = 1.5%)			
Major paraplegia	1 (1.5%)	1 (1.5%)	0 (0%)
Paraparesis	0 (0%)	0 (0%)	0 (0%)
Total (n = 337; 3 operative deaths)			
Major paraplegia	22 (6.6%)	15 (4.5%)	7 (2.1%)
Paraparesis	16 (4.8%)	11 (3.3%)	5 (1.5%)
	38/334 = 11.4%	26 (7.8%)	12 (3.6%)

Table 6. UNIVARIATE ANALYSIS OF VARIABLES EXAMINED FOR ASSOCIATION WITH SPINAL CORD ISCHEMIA

	Spinal Cord Ischemia		
	No (n = 299) n (%)	Yes (n = 38) n (%)	*P* Value (OR; 95% CI)
Preoperative			
Age ≥70 yrs	184 (62)	29 (76)	.11
Male	147 (49)	14 (37)	.17
Serum Cr ≥1.8 mg/dL	45 (15)	5 (13)	1.0
COPD (severe)	47 (16)	6 (16)	1.0
TAA type I-II (n = 151)	125 (42)	26 (68)	.002 (3.0; 1.4–6.8)
Urgent/emergent operation	65 (22)	17 (45)	.004 (2.9; 1.4–6.2)
Rupture	33 (11)	13 (34)	.0004 (4.2; 1.8–9.5)
Dissection	53 (18)	10 (26)	.19
Intraoperative			
Epidural cooling (TAA I-III; n = 271)	161 (69)	19 (51)	.04 (2.1; 0.97–4.5)
Epidural cooling with clamp/sew (I-III; n = 247)	149 (69)	14 (45)	.01 (2.7; 1.3–5.8)
Patent critical zone intercostal (T_9-L1) sacrifice	93 (31)	19 (50)	.03 (2.2; 1.1–4.6)
Intraoperative hypotension	36 (12)	14 (37)	.0003 (4.2; 1.8–9.5)
Operative time (min)*	308.6 ± 95.1	338.7 ± 94.3	.03
Total cross-clamp (min)*	73.0 ± 24.7	86.7 ± 33.5	.02
Visceral cross-clamp (min)*	46.5 ± 14.4	52.6 ± 16.5	.04
Total operative transfusion (cc)*	2,619 ± 1,765	3,978 ± 2,808	.0003
Postoperative			
Reexploration for bleeding	13 (4.3)	4 (10.5)	.17
Renal failure	33 (11)	12 (32)	.002 (3.7; 1.5–8.5)
Cardiac complications	32 (11)	5 (13)	.79
Pulmonary complications	120 (40)	27 (71)	.0004 (3.6; 1.7–8.5)
Stroke	9 (3)	3 (8)	.14

Fisher's exact test used for proportions.
* Wilcoxon (Mann-Whitney) rank-sum test used for continuous variables.
Continuous data: mean ± SD.

increased risk of SCI in types I/II TAA when critical intercostal vessels were oversewn.[15] This important variable is confirmed with multiple studies in the contemporary literature, where the specific influence of intercostal vessel reconstruction has been noted.[7,24–27] Despite the ongoing debate about the worth and/or necessity of this surgical adjunct, the clear-cut weight of evidence in our experience, and corroborated in the literature, is that sacrifice of intercostal vessels in the critical T_9-L1 zone is associated with an increased risk of SCI. Recent angiographic studies in patients with TAA have demonstrated that this is the critical intercostal segment, giving rise to the greater radicular artery in nearly 90% of patients.[28] However, the vagaries of spinal cord circulation are complicated by the fact that in patients with TAA, many or most intercostal vessels are

Table 7. MULTIVARIATE ANALYSIS OF VARIABLES ASSOCIATED WITH SPINAL CORD ISCHEMIA

Variable	OR	95% CI	*P* Value
TAA type I/II vs. type III/IV	2.6	1.1–6.4	.039
Aneurysm rupture	3.5	1.1–11.2	.034
Total cross-clamp time (hrs)	5.2	1.8–14.7	.0019
Patent critical zone intercostal (T_9-L1) sacrifice	2.6	1.1–6.0	.031
Intraoperative hypotension	2.1	1.1–4.0	.028

Model incorporated all factors significant at $P \leq .05$ on univariate analysis.

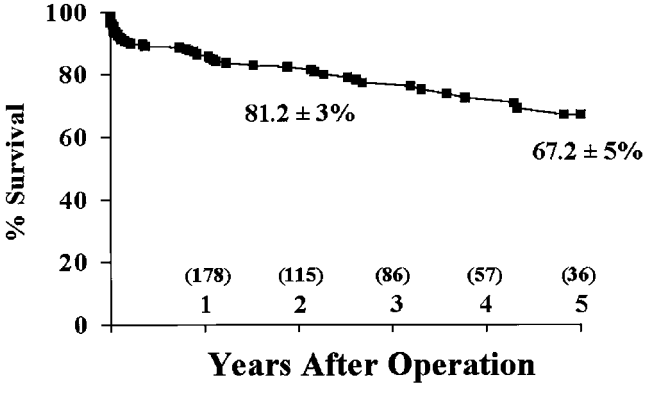

Figure 1. Probability of survival by Kaplan-Meier method after resection of thoracoabdominal aneurysm with survival figures at 2 and 5 years after operation as noted. Numbers in parentheses indicate patients entering corresponding interval.

Table 8. CLINICAL SERIES: THORACOABDOMINAL ANEURYSM REPAIR

Lead Author	Year Published	# Patients	Op. Mortality n (%)	Paraplegia/ Paraparesis	Comment
Svensson[1]	1993	1,509	155 (10)	234 (16)	
Coselli[6]	2000	1,220	93 (7.5)	56 (4.6)	
Schepens[5]	1996	172	18 (10.5)	24 (14.7)	
Grabitz[4]	1996	260	37 (14.2)	39 (15)	
Acher[3]	1998	217	21 (9.7)	17 (7.8)	Desc TA included
Estrera[8]	2001	654	106 (16)	33 (5)	Desc TA included
Jacobs[7]	2002	184	20 (10.8)	5 (2.7)	Types I-III TAA
Van Dongen[9]	2002	118	4 (3.4)	5 (4.2)	Desc TA included ? elective cases only
Present series	2002	337	28 (8.3)	38 (11.4)	
Totals		4,671	482 (10.4)	451 (9.6)	

Desc TA, descending thoracic aortic aneurysm; TAA, thoracoabdominal aortic aneurysm.

previously occluded with mural thrombus such that these patients harbor some degree of antecedent cord collateral circulation. Indeed, Jacobs et al, using intraoperative monitoring of spinal cord blood supply with motor-evoked potentials (MEPs), recently demonstrated (in type I–III TAA) that cord circulation was dependent on lower lumbar arteries or pelvic circulation (i.e., through hypogastric collaterals) in some 24% of patients undergoing TAA repair.[7] Their approach has been to support cord circulation during clamp application by driving distal perfusion pressure to any level to maintain MEPs. The same monitoring is used to guide intercostal reconstruction in various aortic segments (including lumbar arteries) during the course of a sequential clamping technique. Theirs is perhaps the ultimate aggressive posture in intercostal vessel reconstruction, since if MEPs cannot be restored, these authors undertake aortic wall endarterectomy and reconstruct previously occluded (at least at their aortic ostrum) intercostal vessels. As displayed in Table 8, these authors, while reporting an operative mortality exactly equivalent to the mean recorded in the literature, have also recorded perhaps the best results available with respect to the prevention of SCI.[7,25] What remains unclear from their data is the time threshold for reestablishment of cord circulation. In earlier studies using somatosensory evoked potentials, Grabitz et al[4] noted a significantly increased risk of SCI when potentials could not be restored within 20 minutes. The implication, of course, was that intercostal vessel reconstruction was often ineffective as a "stand-alone" strategy, simply because it could not be performed rapidly enough.[4,24] These findings, together with the fact that when cord circulation is dependent on patent T_9-L1 intercostal vessels (which are supported by distal aortic perfusion only during construction of the proximal aortic anastomoses), led us to rely on neuroprotective (rather then distal perfusion) strategies. These methods increase cord ischemic tolerance during the reconstruction, irrespective of the pattern of cord blood supply, until that supply is reestablished, either by intercostal reconstruction or removal of the cross-clamp. With this hypothesis, and founded in convincing experimental literature,[29–34] we developed a clinically applicable technique of regional hypothermic protection for the spinal cord with EC.[12] The moderate levels of hypothermia (mean ≈26°C) achieved in the at-risk thoracolumbar cord with this method have repeatedly been demonstrated to be protective.[29,34] Similar to the results of others reporting longitudinal experience assessing the impact of adjuncts for cord protection,[8] we could demonstrate that EC had a significant impact on SCI when compared to institutional, historical controls.[35] An identical, statistically significant impact on SCI in patients treated for types I-III TAA was noted in the present study. Indeed, not only the incidence but also the clinical impact and severity of SCI were favorably influenced in almost 200 patients managed with EC.[15] Despite such progress, EC has not eliminated SCI, although the immediately occurring, devastating total paraplegia is uncommon in contemporary practice. Frustrating delayed-onset deficits continue to occur in some 10% of extent I-III patients, and although these are often partial and reversible and with acceptable functional outcomes, continued vigilance to perioperative care (especially the avoidance of hypotension) is mandatory. Interestingly, recently presented data from two centers have also emphasized the continued problem of delayed deficits with the adjuncts of distal aortic perfusion and CSF drainage. These authors reported a 10% to 20% mostly delayed deficit rate (including lower-risk isolated thoracic lesions), emphasizing the roles of hemodynamics and CSF drainage as the principal management strategies in the postoperative period.[36,37] We agree that scrupulous attention to avoiding hypotension postoperatively and continued passive CSF drainage are both simply accomplished and important to minimize delayed deficits.[38] Although the role and worth of CSF drainage have been continuously debated since its introduction to clinical use,[39] the recently reported randomized prospective study by Coselli et al has convinced most surgeons of its benefit.[40] Influenced largely by the latter study, our own prac-

tice is to initiate continuous passive CSF drainage in the operating room after restoration of lower extremity perfusion and discontinuation of EC. Drainage is continued for 48 to 72 hours, depending on patient anatomy. For example, patients in whom intercostal vessels were sacrificed are at risk and in our view should have strict control of hemodynamics and CSF drainage for longer periods.

Despite considerable progress, with a diminishing incidence and clinical severity of SCI, no strategy to date has eliminated this complication. As reviewed elsewhere, a multifaceted strategy to minimize the degree and duration of cord ischemia, avoid sacrifice of potential spinal cord blood supply, and prevent the delayed sequela of obligatory intraoperative cord ischemia is likely to produce the best results.[41] With respect to the degree and duration of cord ischemia, minimizing cross-clamp and intercostal ischemic intervals are fundamentals of the operative strategy. We and others[42,43] continue to rely on variants of hypothermia as the best method of cord protection during cross-clamping. Whether the addition of distal perfusion is an important component to lower the degree of cord ischemia via cord collateral circulation is unclear. Many surgeons have adopted this strategy,[7,8,23] and we have used this strategy selectively in patients perceived to be at the highest risk. Unfortunately, the number of patients so managed in the present study was too small to permit firm conclusions; we certainly have observed both SCI and complications specific to left heart bypass such that we employ it in highly selective fashion. As noted above, an aggressive posture of intercostal revascularization has in our view been conclusively proven to be of benefit. Finally, continued focus and research on the prevention of delayed-onset deficits is vital, since a variety of adjuncts and strategies have afforded adequate intraoperative protection.

Despite both considerable perioperative morbidity and the considerable resource expenditure to bring patients through TAA repair, our late survival data are gratifying and continue to compare favorably, for example, to survival after routine abdominal aneurysm repair. Furthermore, consistent with our prior follow-up studies,[2] the majority of operative survivors are returned to their preoperative functional status. Our results compare favorably in this regard to the report of Rectenwald et al, who noted survival of but 67% at 1 year and acceptable functional outcomes in 50% of patients at that interval. Their results were strongly influenced by a perioperative mortality rate of 18% and an additional 15% mortality rate in the first year after operation,[44] whereas actuarial survival at 2 years in the present study was greater than 80%. Finally, the durability of TAA repair in this cohort has been favorable. Recent follow-up studies in our patients indicate that graft-related events such as infections and pseudoaneurysm formation have occurred in but 3% of patients, while 7% will have aortic disease-related events. Typically the latter have been elective resections of aneurysm in other aortic segments.[45]

Despite the favorable impact of EC on SCI over the study interval, we continue to assess strategies to further reduce this complication in those at highest risk. Decreasing the substantial proportion of patients treated in nonelective circumstances will improve overall results.

References

1. Svensson LG, Crawford ES, Hess KR, et al. Experience with 1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg 1993; 17:357–370.
2. Cambria R, Davison JK, Zannetti S, et al. Thoracoabdominal aneurysm repair. Perspectives over a decade with the clamp-and-sew technique. Ann Surg 1997; 226:294–305.
3. Acher CW, Wynn MM, Hoch JR, et al. Cardiac function is a risk factor for paralysis in thoracoabdominal aortic replacement. J Vasc Surg 1998; 27:821–830.
4. Grabitz K, Sandmann W, Stuhmeirer K, et al. The risk of ischemic spinal cord injury in patients undergoing graft replacement for thoracoabdominal aortic aneurysms. J Vasc Surg 1996; 23:230–240.
5. Schepens MA, Dekkar E, Hamnerleynuk RP, et al. Survival and late aortic events after graft replacement for thoracoabdominal aneurysm in 172 patients. J Cardiovasc Surg 1996; 4:713–719.
6. Coselli JS, LeMaire SA, Miller CC III, et al. Mortality and paraplegia after thoracoabdominal aortic aneurysm repair: A risk factor analysis. Ann Thorac Surg 2000; 69:409–414.
7. Jacobs MJ, de Mol BA, Elebass T, et al. Spinal cord blood supply in patients with thoracoabdominal aortic aneurysms. J Vasc Surg 2002; 35:30–37.
8. Estrera AL, Miller CC III, Huynh TTT, et al. Neurologic outcome after thoracic and thoracoabdominal aortic aneurysm repair. Ann Thorac Surg 2001; 72:1225–1231.
9. Van Dongen EP, Schepens MA, Morshuis WM, et al. Thoracic and thoracoabdominal aortic aneurysm repair: Use of evoked potential monitoring of 118 patients. J Vasc Surg 2002; 34:1035–1040.
10. Cambria RP, Giglia J. Prevention of spinal cord ischemic complications after thoracoabdominal aortic surgery. Eur J Vasc Endovasc Surg 1998; 15:96–109.
11. Cambria RP. Thoracoabdominal aortic aneurysm repair: How I do it. Cardiovasc Surg 1999; 7:597–606.
12. Davison J, Cambria R, Vierra D, et al. Epidural cooling for regional spinal cord hypothermia during thoracoabdominal aneurysm repair. J Vasc Surg 1994; 20:304–310.
13. Kashyap VS, Cambria RP, Davison JK, et al. Renal failure after thoracoabdominal aortic surgery. J Vasc Surg 1997; 26:949–957.
14. Cambria RP, Davison JK, Giglia JS, et al. Mesenteric shunting decreases visceral ischemia during thoracoabdominal aneurysm repair. J Vasc Surg 1998; 27:745–749.
15. Cambria RP, Davison JK, Carter C, et al. Epidural cooling for spinal cord protection during thoracoabdominal aneurysm repair: A five-year experience. J Vasc Surg 2000; 31:1093–1102.
16. Huynh TT, Miller CC, Estrera AL, et al. Repair of thoracoabdominal aortic aneurysm in octogenarians. Presented at the 26th Annual Meeting of the Southern Association for Vascular Surgery, Miami, FL, Jan. 17, 2002.
17. Cambria RA, Gloviczki P, Stanton AW, et al. Outcome and expansion rate of 57 thoracoabdominal aortic aneurysms managed nonoperatively. Am J Surg 1995; 170:213–217.
18. Juvonen T, Ergen MA, Galla JD, et al. Prospective study of the natural history of thoracic aortic aneurysm. Ann Thor Surg 1997; 63:1533–1545.
19. Safi HJ, Harlin SA, Miller CC, et al. Predictive factors for acute renal failure in thoracic and thoracoabdominal aortic aneurysm surgery. J Vasc Surg 1996; 24:338–345.
20. Schepens M, Defauw J, Hamerlijnck R, et al. Risk assessment of acute renal failure after thoracoabdominal aortic aneurysm surgery. Ann Surg 1994; 219:400–407.

21. Perko MJ, Norgaard M, Herzog TM, et al. Unoperated aortic aneurysm: A survey of 170 patients. Ann Thorac Surg 1995; 59:1204–1209.
22. Coselli JS, LeMaire SA. Left heart bypass reduces paraplegia rates following thoracoabdominal aneurysm repair. Ann Thorac Surg 1999; 67:1931–1934.
23. Schepens MAAM, Vermeulen FEE, Morshuis WJ, et al. Impact of left heart bypass on the results of thoracoabdominal aortic aneurysm repair. Ann Thorac Surg 1999; 67:1963–1967.
24. Svensson LG, Hess KR, Coselli JS, et al. Influence of segmental arteries, extent and atriofemoral bypass on postoperative paraplegia after thoracoabdominal aortic operations. J Vasc Surg 1994; 20:255–262.
25. Jacobs MJHM, Meylaerts SA, de Haan P, et al. Strategies to prevent neurologic deficit based on motor-evoked potentials in type I and II thoracoabdominal aortic aneurysm repair. J Vasc Surg 1999; 29:48–59.
26. Safi HJ, Miller CC III, Carr C, et al. Importance of intercostal artery reattachment during thoracoabdominal aortic aneurysm repair. J Vasc Surg 1998; 27:58–68.
27. Ross SD, Kron IL, Parrino PE, et al. Preservation of intercostal arteries during thoracoabdominal aortic aneurysm surgery: A retrospective study. J Thorac Cardiovasc Surg 1999; 118:17–25.
28. Kieffer E, Fukui S, Chiras J, et al. Spinal cord arteriography: A safe adjunct before descending thoracic or thoracoabdominal aortic aneurysmectomy. J Vasc Surg 2002; 35:262–268.
29. Marsala M, Vanicky I, Galik J, et al. Panmyelic epidural cooling protects against ischemic spinal cord damage. J Surg Res 1993; 55:21–31.
30. Salzano R, Ellison LH, Altonji PF, et al. Regional deep hypothermia of the spinal cord protects against ischemic injury during thoracic aortic cross-clamping. Ann Thorac Surg 1994; 57:65–70.
31. Rokkas C, Sundaresan S, Shuman TA, et al. Profound systemic hypothermia protects the spinal cord in a primate model of spinal cord ischemia. J Thorac Cardiovasc Surg 1993; 106:1024–1035.
32. Berguer R, Porto J, Fedoronko B, et al. Selective deep hypothermia of the spinal cord prevents paraplegia after aortic cross-clamping in the dog model. J Vasc Surg 1992; 15:62–72.
33. Allen BT, Davis C, Osborne D, et al. Spinal cord ischemia and reperfusion metabolism: the effect of hypothermia. J Vasc Surg 1994; 19:332–340.
34. Martelli E, Cho JS, Mozes G, et al. Epidural cooling for the prevention of ischemic injury to the spinal cord during aortic occlusion in a rabbit model: Determination of the optimal temperature. J Vasc Surg 2002; 35:547–553.
35. Cambria RP, Davison JK, Zannetti S, et al. Clinical experience with epidural cooling for spinal cord protection during thoracic and thoracoabdominal aneurysm repair. J Vasc Surg 1997; 25:234–243.
36. McGarvey M, Hogan MS, Cheung AT, et al. Interventions for reversing delayed-onset postoperative paraplegia after thoracic aortic reconstruction. Presented at the 38th Annual Meeting, The Society of Thoracic Surgeons, February 2002.
37. Manjar HS, Prasad SM, Chu CM, et al. Delayed paraplegia after thoracoabdominal aneurysm repair. Presented at the 38th Annual Meeting, The Society of Thoracic Surgeons, February 2002.
38. Safi HJ, Miller CC, et al. Observations on delayed neurologic deficit after thoracoabdominal aneurysm repair. J Vasc Surg 1997; 26:616–622.
39. Ling E, Arellano R. Systematic overview of the evidence supporting the use of cerebrospinal fluid drainage in thoracoabdominal aneurysm surgery for prevention of paraplegia. Anesthesiology 2000; 93:1115–1122.
40. Coselli JS, LeMaire SA, Koksoy C, et al. Cerebrospinal fluid drainage reduces paraplegia following thoracoabdominal aortic aneurysm repair: Results of a prospective randomized trial. J Vasc Surg 2002; 35:631–639.
41. Svenson LG. New and future approaches for spinal cord protection. Semin Thorac Cardiovasc Surg 1997; 9:206–221.
42. Svensson LG, Hess KR, D'Agostino RS, et al. Reduction of neurologic injury after high-risk thoracoabdominal aortic operation. Ann Thorac Surg 1998; 66:132–138.
43. Kouchoulos NT, Masetti P, Rokkas CK, et al. Safety and efficacy of hypothermic cardiopulmonary bypass and circulatory arrest for operations on the descending thoracic and thoracoabdominal aorta. Ann Thorac Surg 2001; 72:699–708.
44. Rectenwald JE, Huber TS, Martin TD, et al. Functional outcome after thoracoabdominal aneurysm repair. J Vasc Surg 2002; 35:640–647.
45. Clouse WD, Cambria RP, Marone LK, et al. Late aortic and graft-related events after thoracoabdominal aneurysm repair. J Vasc Surg (in press).

Discussion

DR. LOUIS M. MESSINA (San Francisco, CA): Dr. Cambria, I would like to compliment you on these outstanding results, particularly your low perioperative mortality and paraplegia rates. These results were achieved with a very high frequency of emergent repairs, so that makes them particularly laudatory. As you know, paraplegia is devastating both to the patient and to the surgeon, so any technique shown to reduce its frequency as you have deserves careful consideration by any surgeon performing thoracoabdominal aneurysm repair. I had three questions for you.

Since this was a nonconcurrent study, can you exclude the effect of increasing experience, as this was a single-surgeon experience, on reduction in the frequency of paraplegia? For example, were there differences between the two groups in terms of operative time or aortic cross-clamp time?

It wasn't clear to me whether you also utilized lumbar drainage postoperatively, and I would be interested to know whether you employed that technique.

Some groups, including the vascular surgery group at UCSF, use left heart bypass. And in the series utilizing left heart bypass from Texas, spinal cord ischemia and paraplegia has been reported around the 5% range. The Mayo Clinic has not reported a consistently significant reduction in spinal cord ischemia with epidural cooling. I am wondering how you viewed these discrepancies. Are these due to different patient populations?

Finally, I just wanted to mention, as you indicated there was no alternative form of repair of TAAs that surgery, that Tim Chutter and our vascular group at UCSF has performed and published a thoracoabdominal aneurysm repair with a branched endograft.

PRESENTER DR. RICHARD P. CAMBRIA (Boston, MA): I am aware of Tim Chutter's case report of the branched endograft. With respect to CSF drainage, since the recent publication of the prospective study of Coselli and colleagues, we have used this routinely over the past 2 years. With respect to the question on surgeon experience, earlier analysis of our data showed that operative times and cross-clamp times had not changed during the 15-year period over which this data was accumulated.